Stent Fracture and Occlusion after Treatment of Symptomatic Vertebral Artery Ostium Stenosis with a Self-Expanding Device

A Case Report

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Summary

Endovascular stenting with a balloon expandable device is currently the preferred treatment modality for symptomatic proximal vertebral artery stenosis, but high rates of in-stent restenosis remain a major problem, for which stent fracture might be a contributing factor. Limited reports showed that placement of self-expanding stents in the proximal vertebral artery might reduce restenosis; no stent fracture has been reported to date. We describe here a new case of fracture and occlusion of a self-expanding stent after endovascular treatment of symptomatic vertebral artery ostium stenosis.

Introduction

The proximal vertebral artery (VA) is a common site for atherosclerotic stenosis; 20% of patients with symptoms of posterior circulation ischemia have proximal VA occlusive diseases [1]. Despite the lack of sufficient evidence to determine the optimal therapy for proximal VA stenosis, endovascular angioplasty with stenting is currently widely performed with high technical success rates and low periprocedural complication rates, while its long-term efficacy is still uncertain due to high rates of in-stent restenosis (ISR). Drug eluting stents (DES) seem to be able to decrease neointimal hyperplasia related ISR. In a recent system review, a lower restenosis rate of DES (11%) compared to bare metal stents (BMS) (30%) at a mean of 24 months of follow-up was reported for extracranial VA [2]. However, others also described high rates of stent fracture for DES, which might be the major contributing factor to ISR [3,4]. Compared to balloon-expandable stents (BES), self-expanding stents (SES) have enhanced radial expanding force and greater flexibility. Limited reports showed deployment of SES in proximal VA might reduce ISR rates, and no stent fracture has been reported to date. Here we present a new case of stent fracture and occlusion after treating symptomatic VA ostium stenosis with a self-expanding device.

Case Report

A 68-year-old man with diabetes mellitus was admitted with a three-month history of recurrent dizziness and blurred vision despite optimal medical therapy. He had suffered from ischemic stroke ten years before. Duplex ultrasound of carotid arteries demonstrated occlusion of the left internal carotid artery (ICA). Admission cerebral angiography confirmed the left ICA was occluded at its origin, its distal segment and the left middle cerebral artery was reconstituted by the external carotid artery branches via the left ophthalmic artery. The

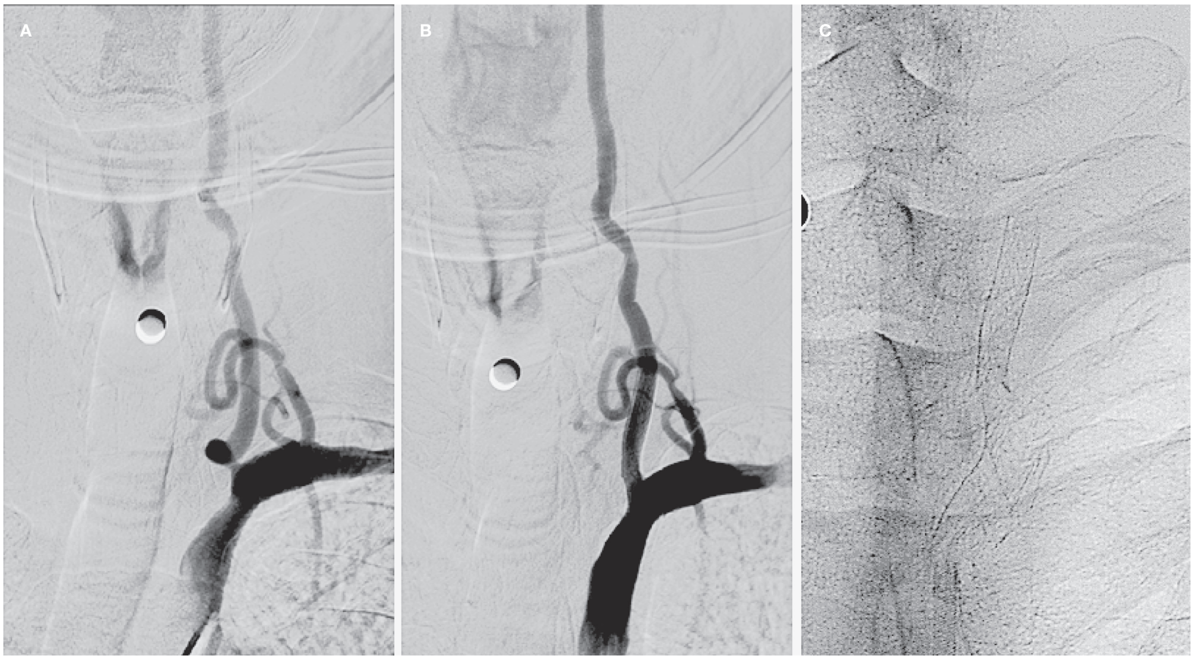

Figure 1 Angiographies of the left SCA and VA pre and postoperatively. A) The left VA had moderate stenosis at its ostium and the proximal V1 segment was tortuous. The SCA proximal to the VA ostium also had mild stenosis. B) The VA ostium stenosis was eliminated after deployment of a tapered self-expanding stent from the distal V1 segment to the proximal SCA, the tortuous proximal V1 segment was straightened and the atherosclerotic plaque of the proximal SCA was also covered. C) No mechanical disruption of the stent wall was observed in the VA.

right VA was occluded at its origin. The left VA showed moderate stenosis at its ostium and the proximal V1 segment was tortuous. The left subclavian artery (SCA) proximal to the VA ostium also had a mild stenosis. We treated the left VA ostium stenosis by balloon angioplasty and stent deployment with a tapered self-expanding device (Acculink 7-10mm/40mm, Abbott Laboratories, Abbott Park, IL, USA). The stent was placed from the mid portion of V1 segment to the proximal SCA, the tortuous proximal V1 segment was mechanically stretched longitudinally and the atherosclerotic plaque of the proximal SCA was covered as well (Figure 1). After the uneventful procedure, the patient received aspirin 100mg/day and clopidogrel 75 mg/day for three months, then aspirin 100mg/day indefinitely.

Ultrasound follow-up four months later showed a decreased blood flow velocity in the left VA, but the patient was asymptomatic until 6 months after the procedure, when he felt recurred mild dizziness. CT angiography (CTA) showed the left V1 segment was occluded from the ostium, and the stent was obviously fractured and kinked at its mid-portion (Figure 2). After a new admission, MRI revealed a new pontine lacunar infarction. Repeated cerebral angiography confirmed total occlusion of the left V1 segment from the origin, the distal VA and the BA were reconstituted via the anastomosis with the left ascending cervical artery and the left occipital artery. The stent fractured due to partial struts intussusception at the portion of previous kink of the V1 segment (Figure 3). We attempted endovascular recanalization of the occluded stent but failed. The patient was discharged with dual antiplatelet therapy and his symptoms disappeared two weeks later.

Discussion

Stent fracture or deformation is not a rare phenomenon in peripheral artery stenting. The incidence of DES fracture in the coronary artery was 0.8-7.7% among which ISR or stent thrombosis occurred at 22-88% [5]. Sfyroeras et al. [6] found a mean 8.9% incidence of carotid stent fracture, which was often associated with ISR but usually asymptomatic. Unlike those lesions, stent deformation or fracture in the proximal VA is seldom reported, and a clear definition is also lacking. The detection rate of VA stent fracture or deformation varies from 0% to 50%, and its relationship to ISR is also ques-

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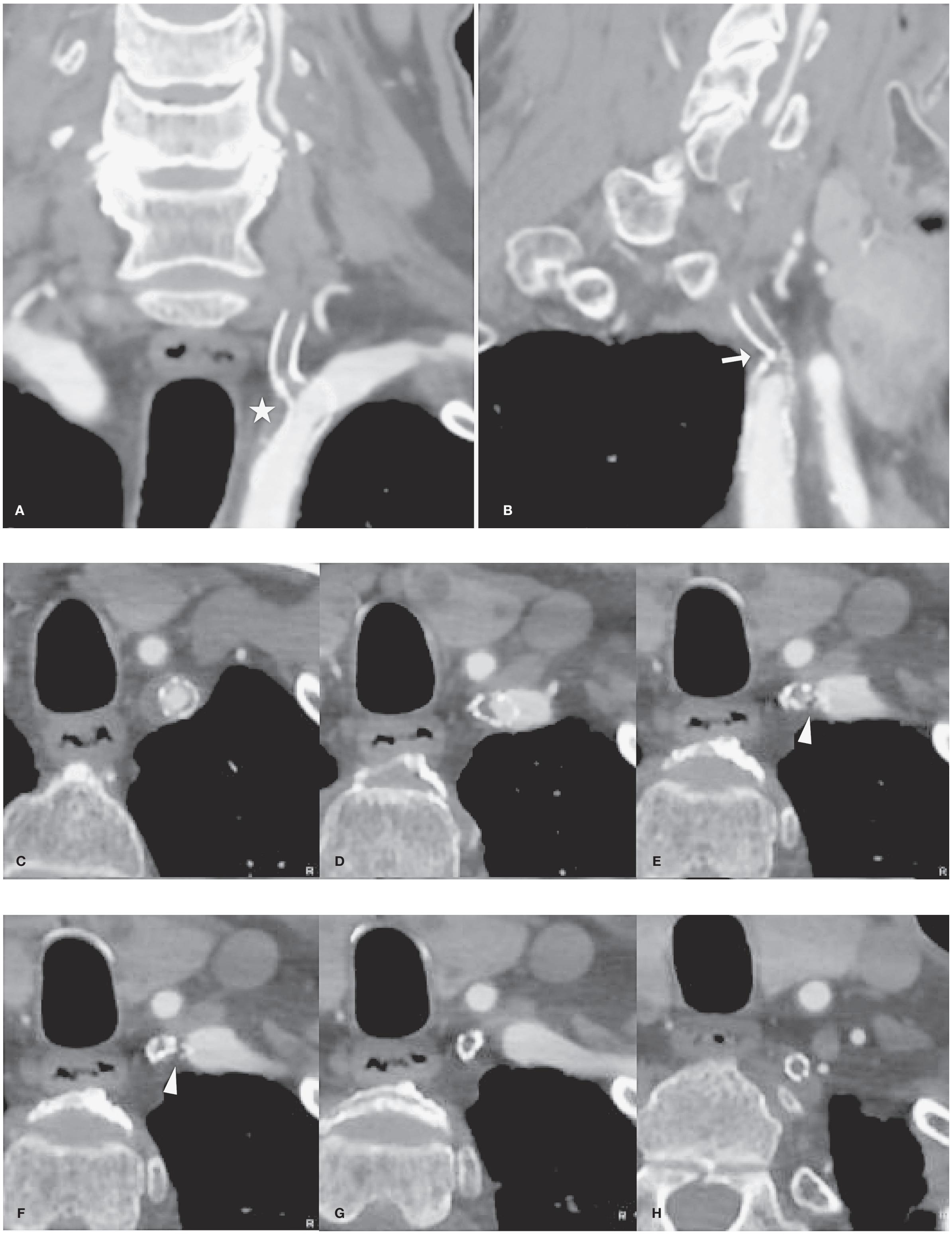

Figure 2 Follow-up CTA. A) MIP image of the posterior-anterior view revealed the left V1 segment was occluded from the ostium (asterisk). B) MIP image of the lateral view demonstrated stent fracture and kinking due to partial struts intussusception and subluxation, and also severe stenosis of the residual in-stent lumen (arrow). C-H) Axial source images of CTA showed disrupted stent struts (arrowhead) and narrowed in-stent lumen.

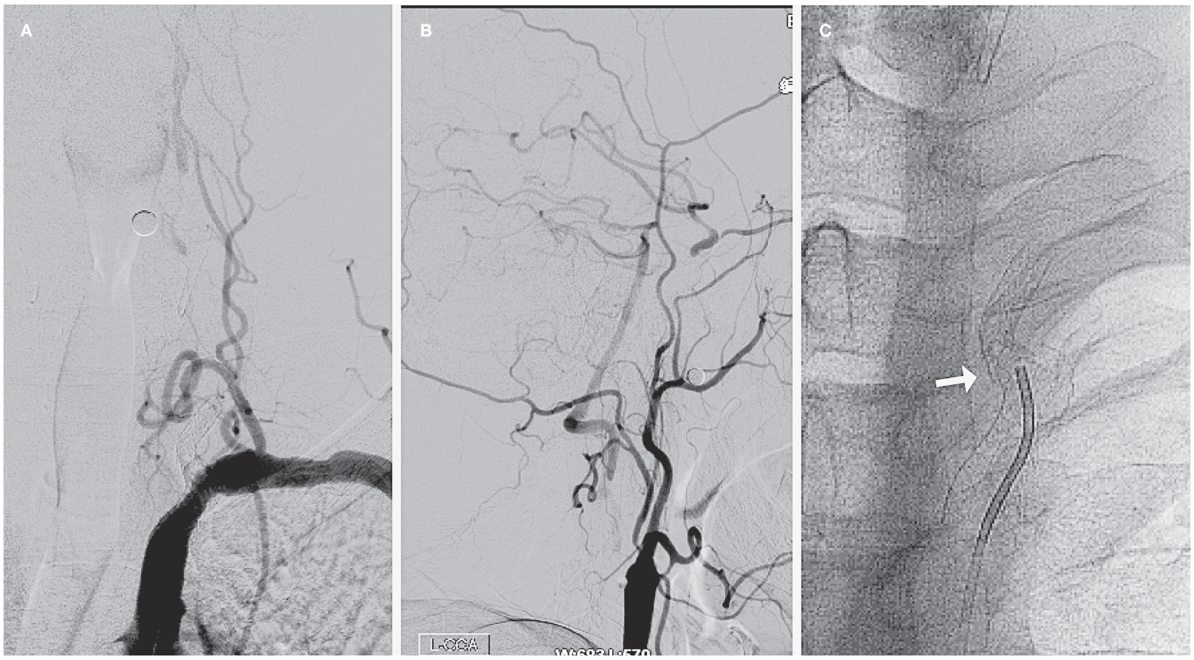

Figure 3 Follow-up angiographies. A,B) Total occlusion of the left V1 segment from the ostium was confirmed, the distal left VA and the BA were reconstituted via the anastomosis with the left ascending cervical artery and the left occipital artery. C) The distal part of the stent was distorted and shortened. Partial stent struts intussusception (arrow) was demonstrated at the portion of previous kink of the V1 segment, but the in-stent lumen seemed patent from the posterior-anterior view.

tionable [4,7-13]. Werner et al.[4] followed up 28 patients who received DES placement in the VA ostium, among whom one fractured stent and 11 compressed or recoiled (up to 50%) stents were detected. They considered stent deformation, not intimal hyperplasia, was the major contributing factor to ISR. Tsutsumi et al.[8] found three stent fractures after 12 VA ostium stenting procedures. All fractures were detected in coronary stents, while none were associated with either recurrent stroke or ISR. Hatano et al.[10] also found all nine coronary stents they deployed were fractured with or without ISR. In addition to ISR, Zhou et al. and Raghuram et al. also described two cases of stent occlusion associated with stent fracture respectively [11-13].

To date, VA stent fracture or deformation occurred in balloon-expandable devices exclusively, none has been reported in self-expanding devices. The reason might be that SES have rarely been applied in the treatment of proximal VA stenosis. Chung et al.[14] treated 20 VA ostium stenoses with SES and no ISR or stent fracture was noted. The main parts of SES were deployed in proximal VA. Li et al.[15] modified the stenting technique and placed SES from the V1 segment to the proximal SCA for 32 cases of VA ostium stenosis, the ISR rate was 3.1% and no stent fracture occurred. SES are considered to be superior to BES for VA ostium stenosis in terms of reducing restenosis as well as stent fracture, especially when placed with that modified technique. SES are designed with more radial force to withstand elastic recoil and recurred compression, their good flexibility and the tapered design can meet the requirement for the diameter gap between the SCA and the VA. Moreover, since atherosclerotic plaque formation often began in the SCA and extended a few centimeters into the proximal VA and restenosis occurred significantly more often in those with an ipsilateral SCA stenosis, modified deployment of SES can theoretically treat both lesions simultaneously.

Here, we present a case of fracture and occlusion of a self-expanding stent after treating symptomatic VA ostium stenosis. We suppose the long stent deployed with the modified technique might be the main contributing factor for stent fracture in our case, the VA occlusion was caused by stent fracture and thrombosis. Neck and head movement can create flexion, stretching and torsion forces to the stent in proximal VA, making hinge points and can result in mechanical fatigue and fracture. According to the law of the lever, this impact can be much stronger after a long stent was placed with one half in the V1 segment and the other half in the proximal SCA, especially when there is a sharp angulation between VA and SCA. Moreover, the proximal V1 segment of our case was significantly tortuous, and stretching it by a stent could generate a force tending to return it to the original state, which can also deform and break the stent. Similar conditions have been proved in coronary artery where excessive tortuosity, angulation of coronary artery or change in angulation after stent implantation were risk factors for stent fracture.

In the previous literature, deformation or fracture of VA stents were all detected by angiography or simple radiogram. In the present case, we also observed struts intussusception and stent shortening, but the residual lumen of the stent seemed patent. The reason might be that the tortuosity of the proximal V1 segment and the kink of the stent were mainly in the dorsal sagittal plane. Lateral view of the stent from angiography was impeded by the overlying bony structures. We supposed some VA stent fractures in previous reports might also have been overlooked for that reason. In our case, MIP images of CTA clearly revealed discontinuity and kink of the stent from the lateral view, especially severe stenosis of the residual in-stent lumen and partial struts intussusception and subluxation. From the axial source images of CTA, we also found the deformed stent shape and narrowed in-stent lumen. Considering a tortuous V1 segment is not unusual in the stenting procedures for proximal VA stenosis, we think CTA might be a good alternative to detect stent fracture or deformation.

Conclusion

Stent fracture can be detected from SES when treating proximal VA stenosis, and should be kept in mind since it may cause stent occlusion. More angiography or CTA follow-up studies are warranted to investigate the incidence and clinical implications of stent fracture.

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Editorial Comment

This interesting paper illustrates that an elegant technique can lead to a poor result when it is not used correctly. The subclavian artery and vertebral artery are dynamically different from the carotid artery. For a long time we have known that self-expanding stents, which are so adequate in carotid artery treatments, frequently generate myo-intimal hyperplasia in subclavian artery stentings. This is also true for vertebral artery stenosis. Balloon expanding stents remain, in our opinion, much better.

The second point is the diameter of the stent which was used. For many years, we personally had overdilated origins of vertebral arteries with too large diameter balloon expandable stents (5 mm). We think that a vertebral artery is a very reactive artery that should not be dilated than 4 mm for the largest stents. It is true that self-expanding stents adapt their diameters as we do for distal cervical carotid arteries stentings but 7mm stent diameter stenting probably causes too much stress on a very reactive vertebral artery. Actually stent modification was immediate and can be seen on the angiography control the first day (Figure 1C). This led to occlusion of the artery.

The main difficulty is to treat stenosis at the origin of a vertebral artery completely with a balloon expandable stent knowing that the ostium of the vertebral artery is located on the posterior wall of the subclavian artery. Good stenting with a balloon expandable stent should lead to a picture of the origin of the stent "hanging" in the subclavian artery on angiography control. An associated subclavian artery at the same level would have been treated previously by simple balloon angioplasty without a stent using a balloon widely covering the origin of the vertebral artery. This will not lead to occlusion of the vertebral artery which is only pushed upward during dilation.

Jacques Théron